of chest-pain within 72 hours, or ongoing chest-pain, were included. ECG criteria to be fullfilled was transient or persisting ST-depression and/or T-wave inversion. Subcutaneos injections of dalteparin 120 IU/kg were given twice daily the first 6 days followed by 7500 IU once daily the following 35–45 days. P-fibrinogen (Fib.) and P-CRP were measured at inclusion. Event rates (%) during the 150 days follow-up period were evaluated in relation to median levels of fibrinogen and CRP.

Conclusion: In patients with unstable coronary artery disease initially treated with dalteparin, and long term treatment with aspirin, elevation of fibrinogen and CRP at admission indicates an increased risk of subsequent acute myocardial infarction and death. The importance of inflammation as a pathological component in unstable coronary artery syndromes needs further evaluation.

2:30

793-3 C-Reactive Protein in Unstable Angina: Lack of Association With Ischemic Activity or Complex Lesion Morphology

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C-reactive protein (CRP), a sensitive marker of inflammation, is elevated in unstable angina (UA) although it is not known whether a high CRP relates to ischaemic activity or complex lesion morphology (CLM). CRP levels were measured, (Beckman turbidimetric method), in 72 pts (53 males), mean age 63.6 (46–79) yrs, with refractory UA (Braunwald class IIIB). Transient myocardial ischemia (TMI) was detected using continuous ST segment monitoring and angiographic analysis was performed by two observers blinded to other study data. Median CRP was 0.89 mg/dl (0.48–20.75), and was elevated (> 1 mg/dl) in 32 (44%) pts. TMI was present in 20 (28%) pts despite maximal medical therapy. 14 (19%) pts had 1 vessel disease, 22 (31%) 2 vessel, and 36 (50%) 3 vessel disease. CLM was present in 35 (49%) pts and thrombus in 7 (10%). Pts with TMI had similar CRP levels and was raised in a similar proportion (see table). Pts with CLM had similar CRP levels, although CRP was higher in pts with multi-vessel disease ($P < 0.05$). CRP was raised in 4/7 (57%) pts with thrombus.

CRP	TMI	No TMI	CLM	No CLM	1 VD	2 VD	3 VD
> 1 mg/dl (%)	8 (40%)	24 (46%)	15 (43%)	27 (46%)	4 (29%)	7 (32%)	21 (58%)
Median (range)	0.8 0.5–21	0.95 0.5–7.9	0.74 0.5–21	0.99 0.5–7.9	0.69 0.5–7.9	0.68 0.5–21	1.12 0.5–20

Conclusion Although CRP is elevated in refractory unstable angina, and is associated with multi-vessel disease, it does not predict ischemic activity or angiographic markers of acute plaque rupture.

2:45

793-4 Inflammation & Thrombosis in Unstable Angina. Insights From Directional Coronary Atherectomy Tissue Analyses

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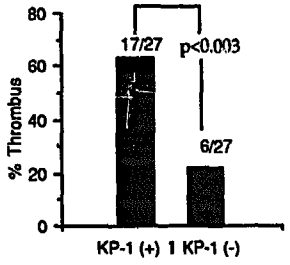
Plaque disruption with thrombus (T) is the major pathogenetic mechanism for the acute coronary syndromes of myocardial infarction and unstable angina (UA). Recently, clinical studies have suggested a role for inflammation in UA. The relationship between inflammation and thrombus in UA has not been well studied. Analysis of tissue from directional atherectomy specimens can be used to correlate inflammation and thrombus. Thus, we analyzed the results of immunohistochemical staining for macrophages (KP-1) in 54 culprit lesions (19 with rest angina, 15 with new onset/crescendo, 10 post-infarction and 10 with stable angina. Thrombus was detected by H and E staining. KP-1 staining was graded as 0 to 2+ and 2+ was considered positive(+). Pathologic specimens were analyzed blindly.



Thrombus was found predominantly in rest angina and post-Infarction. KP-1 staining was more common in all syndromes other than stable. A positive KP-1 was significantly associated with thrombus particularly in rest angina and post-infarction where 14 of 17 KP-1 positive lesions were positive for thrombus. Thus, these data suggest a strong association between inflammation and thrombus in unstable rest angina and post-infarction.

3:00

793-5 Measurement of Leukocyte Sequestration and Adhesion Molecule Expression in the Coronary Circulation of Unstable Angina Patients

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Polymorphonuclear leukocyte, PMN, adhesion and activation has been demonstrated to be of great importance in the mechanisms of myocardial ischemia and reperfusion injury. Evidence from animal models demonstrates that upon activation by inflammatory mediators, PMNs are trapped in the coronary circulation. Despite the important role of PMNs in postischemic injury, little is known concerning the accumulation of these cells in the heart after interaction with unstable plaques. This study evaluated the magnitude of PMN sequestration in the coronary circulation of unstable angina (UA) patients and the changes in the expression of adhesion molecules caused by interaction with active lesions. Ten UA patients undergoing catheterization within the first 48 hours of symptoms and four control patients (Ct) had blood samples collected simultaneously from the aorta (Ao) and coronary sinus (CS) prior to the injection of contrast medium. PMN counts revealed a small but significant decrease in the number of cells collected in the CS compared to the Ao ($p < 0.05$). PMNs also had typical L-selectin (activation associated) shedding ($p = 0.001$). This pattern of activation, however, was not paralleled by CD11b and CD18 adhesion molecules, suggesting that cells which had this integrin upregulation remained sequestered due to adhesion to the coronary endothelium.

Group	Coronary Sinus			
	Cell Count	*L-selectin*	*CD11b*	*CD18*
UA (n = 10)	0.93 ± 0.03*	0.79 ± 0.05**	1.04 ± 0.12	0.88 ± 0.1
Ct (n = 4)	0.99 ± 0.03	1.04 ± 0.01	1.00 ± 0.05	0.98 ± 0.03

Results expressed as % of control (Ao measurements); *p < 0.05, **p = 0.001

Thus, in unstable angina L-selectin shedding and PMN accumulation occurs within the coronary circulation which may in turn contribute to further vascular or myocardial damage.

3:15

793-6 Plasminogen Activation in Unstable Angina Is Associated With an Acute Phase Response but Not With Activation of the Hemostastic System

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Coronary thrombosis is the major pathogenetic event leading to ischemia in pts with unstable angina (UA). This event involves the coagulation and the fibrinolytic systems, and, possibly, the inflammatory system. To assess the role of activation of fibrynolytic system and its relation with the hemostatic and the inflammatory systems, we measured plasma levels of Plasmin-α2-antiplasmin (PAP), C-Reactive protein (CRP) and Thrombin-AntiThrombin III (TAT), as markers of plasminogen activation, inflammation and thrombin production, respectively. We also measured plasma levels of D-Dimer (DD), a fibrin degradation product, to assess actual lysis of fibrin. We studied 32 pts admitted to our CCU for severe UA. Blood samples were taken at CCU admission. As controls(C) we studied 20 healthy volunteers. *Results* (median and range): Elevated levels were considered above mean +2 SD for PAP (700 ng/ml), DD (70 μg/l) and TAT (4 μg/ml) and levels > 3 mg/l for CRP (90°percentile of healthy subjects). Elevated levels of PAP were observed in 16/32 pts (50%), but elevated levels of DD and TAT were found only in 5/32 pts (16%). Conversely CRP was raised in 23/32 pts (71.8%). PAP levels were significantly higher in pts with UA than in C (698 ng/ml, range 268–1899 vs 413 ng/ml, range 201–826; $p = 0.017$) but they did not correlate with levels of DD (20.3 μg/l, range 5.7–246; $r = 0.06$, p = ns) or of TAT (2.1 μg/ml, range 0.95–23, $r = -0.05$, p = ns). Conversely a significant correlation was observed between PAP and CRP (4.55 mg/l, range 0.7–82; $r = 0.47$, $p = 0.005$). *Conclusions:* In pts with UA there is a